

Clinical Section

Anterior Poliomyelitis

The first meeting of the Winnipeg Medical Society for the season, held on September 18th, was given over to a symposium on Anterior Poliomyelitis. Following the various papers, the meeting was opened for general discussion. The President, W. E. Campbell, B.A., M.D. (Man.), was in the chair.

Clinical Features

By

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The purpose of this meeting tonight is to review the facts concerning the present outbreak of poliomyelitis, to re-emphasize the value of convalescent serum in the preparalytic stage, and to discuss the prevention, control, and the early recognition of the disease. Whatever I may have to say will be of a very general nature and refers merely to my experience during the epidemic of 1928, when I was privileged to assist the staff of King George Hospital in recording the case histories of patients admitted to that institution.

J. E. Gordon has stated that the diagnosis of preparalytic poliomyelitis consists of "**a healthy clinical suspicion and a lumbar puncture.**" The prompt suspicion of the disease from clinical evidence is the task that confronts every practitioner during an epidemic.

Experimental evidence strongly suggests that poliomyelitis is entirely neurotropic—that the virus travels from the nasopharynx through the cribriform plates, along the olfactory nerves to the central nervous system where it is propagated along nerve tracts. However, the early clinical history of the disease suggests an *initial systemic reaction* marked by constitutional symptoms such as fever, malaise, gastro-intestinal upsets with vomiting, diarrhoea or constipation. This is quickly followed by the *preparalytic stage proper* with symptoms due to invasion of the central nervous system—headache, irritability or drowsiness, hyperaesthesia, pain on spinal flexion, changes in reflexes, etc. The *final state of paralysis* usually occurs on the second or third day after the onset—except in the so-called "*dromedary*" type where the initial symptoms clear up entirely and then several days later re-appear with onset of paralysis. Undoubtedly many *abortive cases* also occur during an epidemic which frequently pass unnoticed. One must also mention the *fulminant type* with rapid bulbar involvement and death which seem prone to occur during the earlier stages of epidemics.

In the common spinal form the earliest symptoms are not characteristic. The child feels out of sorts and often peevish and disinclined to play, fever is the rule—generally moderate 100°-102°, appetite is lost and vomiting may occur with some looseness of the bowels. Frontal headache occurs early in most cases, and is followed by *painful stiffness of the neck* and spine so that the child resents any movement. This gives rise to the very valuable "*spine sign.*" The child is often unable to sit upright without supporting himself on his arms and assumes a rather erect, proud position. Attempting to flex the spine and neck to bring the head between the knees demonstrates rigidity of the spine and elicits pain in the neck and in lumbar region. A weakly positive Kernig's sign may sometimes be found. Early in the course of the disease the *reflexes* may be hyperactive but later are generally diminished, especially the superficial reflexes; the tendon reflexes are generally asymmetrically involved. A rather coarse ataxic *tremor* may be evident whenever the child moves. Twitching of various muscle groups may be observed, especially during sleep. Many observers report a characteristic expression during the early stages, described as a "dazed look"—"a vague apprehension," etc.

The onset of fever and constitutional symptoms associated with headache, rigid spine and neck calls for the performance of *lumbar puncture*. There may possibly be some slight danger of initiating involvement of the central nervous system if lumbar puncture is performed too early, but such danger seems rather remote in contrast to the value of C.S.F. examination for establishing a definite diagnosis. The fluid is usually clear or faintly opalescent, under moderately increased pressure (150-200 mm. water). The cell count is increased usually from 15-500, with an average of about 25-40 predominantly lymphocytes, except in the very early stages when polymorphs may occur. A slight excess of globulin appears usually on the second or third day. Sugar and chlorides are usually normal in poliomyelitis in contrast with the usual increased sugar of epidemic encephalitis and the decrease chloride content of tuberculosis meningitis. A weak mid-zone reaction is usually evident in the colloidal gold curve. Lumbar puncture is of great value not only in establishing a definite diagnosis of poliomyelitis but also in differentiating the condition from meningococcal meningitis, encephalitis lethargica, and tuberculous meningitis. It also serves to differentiate poliomyelitis from diseases with an intact nervous system and thus save a waste of convalescent serum. There is a tendency during an epidemic to inject serum into every child exhibiting constitutional symptoms, or pain in the extremities; thus, cases of osteomyelitis, acute rheumatic fever, etc., may be wrongly diagnosed. A careful consideration

of the history, physical signs, and, if necessary, examination of spinal fluid, will usually prevent mistaken diagnosis.

The question of administering convalescent serum to cases in whom paralysis has already set in is a point for discussion. It is doubtful whether the late administration has much effect on the degree of paralysis, but a distinct amelioration of general symptoms is usually seen. Some patients during the preparalytic stage complain of a generalized paresis which does not necessarily mean irreparable involvement of anterior horn cells, and such patient should not have the benefit of serum withheld.

A Brief Review of the 1928 Epidemic

By

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My first experience with epidemic poliomyelitis was in 1926 when as resident pathologist under Professor Wm. Boyd I had the opportunity of studying 6 cases of a very peculiar type. The striking feature of these cases was the common occurrence of bulbar paralysis with no sign of involvement of the limbs. A number of these children died of acute oedema of the lungs.

In 1928 the Medical Research Committee under the chairmanship of Professor C. R. Gilmour appointed a number of us to act as Honorary Consultants to aid in the diagnosis and treatment of the disease. A brief review of this epidemic should be of interest at the present time. Four hundred and thirty five cases were reported throughout the province between July 1st and November 15th during that year.

The following table compiled by Professor A. T. Cameron shows the incidence of the disease by months. It will be seen that the peak of the epidemic occurred in September. In fact in the city the peak load of cases occurred on September 4th.

| TABLE I | | | | |
|--------------------------|----------|---------|------------------|-------|
| Month | Winnipeg | Suburbs | Rest of Province | Total |
| July | 14 | 4 | 3 | 21 |
| August | 87 | 24 | 32 | 143 |
| September | 112 | 34 | 85 | 231 |
| October | 22 | 4 | 9 | 35 |
| November (to 15th) | 0 | 1 | 4 | 5 |
| Totals | 235 | 67 | 133 | 435 |

The daily records suggested an increased incidence every 5 to 7 days. The incidence of age and sex is shown in Tables II and III.

| TABLE II | | | | | | | | |
|-----------------------|----------|-------|---------|-----|------------------|-------|-------|-------|
| INCIDENCE OF AGE | | | | | | | | |
| | Winnipeg | | Suburbs | | Rest of Province | | Total | |
| | No. | % | No. | % | No. | % | No. | % |
| Less than 5 | 76 | 32.6 | 22 | 34 | 32 | 24.8 | 130 | 30.5 |
| 5 to less than 10 .. | 82 | 35.2 | 31 | 48 | 38 | 29.5 | 151 | 35.4 |
| 10 to less than 15 .. | 40 | 17.2 | 8 | 13 | 29 | 22.5 | 77 | 18.1 |
| 15 to less than 20 .. | 22 | 9.5 | 3 | 5 | 18 | 14.0 | 43 | 10.1 |
| 20 to less than 25 .. | 8 | 3.4 | 0 | 0 | 4 | 3.1 | 12 | 2.8 |
| 25 and over | 5 | 2.1 | 0 | 0 | 8 | 6.1 | 13 | 3.1 |
| Totals | 233 | 100.0 | 64 | 100 | 129 | 100.0 | 426 | 100.0 |

| TABLE III | | |
|------------------|-----|-------|
| Incidence of Sex | | |
| No. of males | 233 | 55.7% |
| No. of females | 185 | 44.3% |

It is interesting to note here that the disease is affecting children of lower age during the present epidemic suggesting that the older children acquired immunity during the 1928 epidemic. It is also interesting to note that there were no cases from Boissevain where very heavy infection has occurred this year.

Of the 37 deaths which occurred in this epidemic 17 occurred outside the city.

Convalescent serum was used extensively during the epidemic under the direction of Professor F. T. Cadham. Over 8,000 c.c. was issued during the epidemic. The ratio of serum treated to reported cases was very high during September.

One hundred and sixty one cases were specially studied during the epidemic and careful records kept. Of these 161 cases 74 received serum in the preparalytic stage of the disease, 54 received no serum and 33 received serum after paralysis had occurred.

The results of serum therapy are summarized in Table IV.

| TABLE IV | | | | | | | |
|-----------|--------------|--------------------------|------------------------|-----------------------------------|----------------------------|--------|----|
| Group | No. of Cases | No. Completely Recovered | | Number Showing Residual Paralysis | Percent Residual Paralysis | Deaths | |
| | | Recov-ered | Com-pletely Recov-ered | | | No. | % |
| I | 57 | 53 | 93 | 4 | 7 | 0 | 0 |
| II | 17 | 16 | 94 | 1 | 6 | 0 | 0 |
| III | 33 | 7 | 22 | 15 | 45 | 11 | 33 |
| IV | 54 | 14 | 26 | 34 | 63 | 6 | 11 |

Group I—1 dose intramuscular serum in pre-paralytic stage.

Group II—2 or more doses of serum by various routes (pre-paralytic stage).

Group III—Serum given after onset of paralysis.

Group IV—No serum given.

Granted that the virus becomes attenuated in the later stages of an epidemic, in order to prove conclusively that serum is effective it becomes necessary to compare the figures during one given month of the epidemic. That there is such a decrease in severity is shown in Table V.

TABLE V

| | Number of Cases | Early Para- lysis % | Residual Para- lysis % | Deaths % | Com- plete Re- covery % |
|--|-----------------------|------------------------------|---------------------------------|-------------|-------------------------------------|
| August— | | | | | |
| Cases without serum treatment | 28 | 96 | 68 | 18 | 14 |
| September— | | | | | |
| Cases without serum treatment | 22 | 82 | 59 | 5 | 36 |

If, however, we compare the results of serum treatment with those of the controls, for the month of September, we find that the decrease in virulence indicated in Table V is much less evident than the improvement indicated by the figures for treated cases shown in Table VI. For example, of the 22 cases in September who received no treatment 36 per cent made a complete recovery, while of 50 cases in the same month who received serum in the pre-paralytic stage 94 per cent made a complete recovery.

TABLE VI

| | Number of Cases | Early Para- lysis % | Residual Para- lysis % | Deaths % | Com- plete Re- covery % |
|---------------------------------------|-----------------------|------------------------------|---------------------------------|-------------|-------------------------------------|
| September— | | | | | |
| No serum | 22 | 82 | 59 | 5 | 36 |
| September— | | | | | |
| Serum in pre-paralytic stage | 50 | 10 | 6 | 0 | 94 |

It is generally recognised that the effect of the virus is greater in the country than in the city—that cases are more severe in the rural districts. If our control cases were entirely from the country and the treated cases from the city our results would be open to criticism. In Table VII the September cases are further divided into those from the city, and those from the country.

TABLE VII
CITY CASES IN SEPTEMBER

| | Number of Cases | Early Para- lysis % | Residual Para- lysis % | Deaths | Com- plete Re- covery |
|---------------------------------------|-----------------------|------------------------------|---------------------------------|--------|--------------------------------|
| No serum | 14 | 86 | 50 | 7 | 43 |
| Serum in pre-paralytic stage | 43 | 7 | 5 | 0 | 95 |

COUNTRY CASES IN SEPTEMBER

| | Number of Cases | Early Para- lysis % | Residual Para- lysis % | Deaths | Com- plete Re- covery |
|---------------------------------------|-----------------------|------------------------------|---------------------------------|--------|--------------------------------|
| No. serum | 8 | 75 | 75 | 0 | 25 |
| Serum in pre-paralytic stage | 7 | 29 | 14 | 0 | 86 |

A final criticism may be raised: Are not the beneficial results observed in the serum-treated cases due to the fact that they are milder cases, that the controls for the most part are those seen after paralysis had occurred? This objection is not so easily disposed of, but we may answer it from several different angles.

First, the marked actual differences in percentages of complete recovery between the treated cases and the controls in September should in themselves be sufficient to override this objection.

Secondly, during that month nearly every case occurring in the city was seen by the Committee, and there is no reason to suppose that the severe cases were almost entirely absent from the treated group of nearly 60 cases.

Thirdly, if we assume that serum has no therapeutic value whatever, an accurate analysis according to the statistical method by a qualified statistician has shown that the results shown in Table V. could only occur once in five hundred times. The arbitrary level of significance is taken to be one in thirty.

Fourthly, following Draper's view that a cell count of over 100 indicates a serious type of disease (8), a comparison of the cell counts in treated cases and controls in Table VI. shows no appreciable difference in numerical incidence of the counts from which it would follow that there is no significant difference in the average severity of the two groups of cases. 92% of these counts in the treated cases were made by the fifth day of the disease. 82% were made by the fifth day in the controls. The difference of 10% is not felt to be significant as there is no marked change in the cell counts during the first week of the disease. (Peabody 5).

TABLE VIII
INCIDENCE OF CELL COUNTS IN TREATED
AND CONTROL CASES

| Cell Counts | Treated Cases | Control Cases |
|-----------------|---------------|---------------|
| 10- 99 | 36 | 23 |
| 100- 199 | 12 | 10 |
| 200- 299 | 6 | 4 |
| 300- 399 | 0 | 0 |
| 400- 499 | 2 | 1 |
| 500-1000 | 2 | 2 |
| 1000-2000 | 2 | 0 |
| Total | 60 | 40 |

Finally, it is felt that a comparison of the results in cases which had early paresis or paralysis with the end results will give us the true index of recovery, in treated and untreated cases. It is well known that the initial lesion is the ultimate reaction to the virus. For example, no case which initially developed paresis without paralysis, subsequently—that is after a lapse of three or four weeks—developed paralysis. The results of the comparison are seen in Tables VII and VIII.

TABLE IX
END RESULTS IN CASES WHICH HAD EARLY
PARESIS OR PARALYSIS

| | Number of Cases | Percent Recovered | Percent Residual Paralysis or Death |
|---------------------------------|-----------------------|----------------------|--|
| Treated Groups I and II | 13 | 62 | 38 |
| Control Groups III and IV | 82 | 20 | 80 |

TABLE X
RESULTS IN CASES WHICH HAD EARLY
PARESIS ALONE

| | Number Cases | Percent Recovered | Percent Residual Paralysis |
|---------------------------------|-----------------|----------------------|----------------------------------|
| Treated Groups I and II | 12 | 67 | 33 |
| Control Groups III and IV | 30 | 40 | 60 |

These results seem to justify the use of serum in the pre-paralytic stage.

IMMEDIATE RESULTS FOLLOWING
THE USE OF THE SERUM

Within a few hours following the use of the serum the usual result was a drop in temperature and complete recovery from most of the symptoms complained of. It should be noted, however, that a drop in temperature and apparent beneficial results following the use of the serum occurred in cases afterwards proved to be ordinarily febrile disturbances and not poliomyelitis. This, of course, does not detract from the therapeutic value of convalescent serum in poliomyelitis, but indicates that some part of the beneficial effect may be due to other than specific factors in the serum.

CONCLUSIONS

1. Convalescent serum is of value when administered in the pre-paralytic stage of the disease.

2. The intramuscular route of administration is simple, safe, and sufficiently efficacious to justify its use during an epidemic.

(Associated in this work were Drs. Gilmour, Cadham, A. T. Cameron, B. Chown, L. G. Bell and Mary MacKenzie).

**Report of Poliomyelitis Outbreak
in Morton Municipality, 1936**

By

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Eight deaths and eight cases of residual paralysis in Morton Municipality since June 18th have tragically exemplified the description by Simon Flexner that poliomyelitis is "one of the saddest of diseases." The picture, however, has its brighter side also, as there have been over 30 cases in which complete recovery has occurred. My remarks, as far as statistics are concerned, will be based entirely on the cases occurring in Morton Municipality, although opinions expressed have been formed from studying over 75 cases throughout the province during the summer.

INCIDENCE

General Incidence. An approximate estimate of the incidence among a general population is impossible because a considerable number of mild cases are either never recognized or never notified. The rural Municipality of Morton, including the town of Boissevain, has a population of 2,666, and since June 18th there have occurred 47 cases, giving a

case incidence of 17.3 per 1,000. This is not taking into account several additional cases where serum was administered, but on which diagnosis was unconfirmed. This extremely high rate may be accounted for by three factors: (1) a highly susceptible population, no cases having occurred here for over 20 years; (2) the particularly high virulence of the virus in this epidemic. This is borne out by many instances, and particularly should it be noted that of the first six cases, three died and three have residual paralysis. The first eight cases all occurred within an area of 6 miles diameter, showing it to be an outbreak of great intensity. (3) Possibility for wide dissemination of the virus by contact existed for the first few weeks after the initial outbreak. Altogether, 31 cases occurred in the country and 16 in the town, giving a case incidence in the rural sections of 16.2 per 1,000 as against 19.3 in the town.

It may be of interest to observe the incidence of cases by weeks during the epidemic. It lasted altogether 12 weeks. During the first week there were two cases with a five day interval between. Then there was an interval of 16 days until the third case occurred, and following this, a further interval of 10 days until the fourth case. This brings us to the fifth week. Now there was a sudden outbreak of 8 cases occurring in four days. This was followed by a five day interval of no cases, and in the next week there were 4 cases. For the next three weeks the period of greatest intensity occurred, 11 cases in the seventh week, 11 in the eighth week, and 7 in the ninth week. Then there was, as is usual, the almost abrupt fall of incidence, only one case occurring in each of the three succeeding weeks, and these all of a very mild nature.

Sex played no appreciable factor in the outbreak, cases being equally divided amongst the male and female. **Age**, however, was of greater significance, and was felt to have an important influence on the fatality rate. It has been proven that the younger the age of the patient attacked with poliomyelitis, the better the chance for survival. (1) In this series of cases the ages varied between 3 years and 47 years. The average of males was 16.1 and of females, 15.9, a much higher average than is usual. An analysis of age groups shows the following:—

| | | Morton 1936 | Manitoba 1928 |
|-------------------------|----------|----------------|------------------|
| Less than 5 years | 3 cases | 6.4% | 30.5% |
| 5 to 10 years | 9 cases | 19.5% | 35.4% |
| 10 to 15 years | 9 cases | 19.5% | 18.1% |
| 15 to 20 years | 13 cases | 28.2% | 10.1% |
| 20 to 25 years | 4 cases | 8.9% | 2.8% |
| Over 25 years | 8 cases | 17.4% | 3.1% |

It will be seen that the greatest age incidence occurred in the 15-20 group, and that nearly 50% of cases occurred between the ages of 10 and 20. The comparison with the age groups affected in the province during the epidemic of 1928 shows a striking comparison, 66% of these cases occurring under the age of ten years. This high age incidence in the present outbreak, would also account for the high mortality rate.

MORTALITY

There have been eight deaths, giving a case fatality rate of 17.3%, this also proving the virulence of the infecting organism. Eight cases had a residual paralysis, leaving a complete recovery of 65.4%.

INCUBATION PERIOD

Contact was tracable in 29 of these cases. In the remaining 18 cases, no such history could be elicited. The interval between known contact and onset of symptoms varied between four days and nineteen days, with an average for the total of 10.6 days. However, in some instances, we were able to get very definite information. For instance, 7 cases occurred where two in the one family contracted the infection. In two of these instances the illness occurred on the same day within a few hours of each other, obviously having contracted infection from a common source. Of the other five, one occurred seven days following the first case, three occurred eight days later, and one on the ninth day. The families were, of course, in quarantine and therefore no outside source of contact was possible. Altogether we have eight proven cases where onset of symptoms occurred on the seventh or eighth day following contact or exposure, and this we take to be the nearest approach to accuracy in the matter of incubation period.

TRANSMISSION

It is probable that an epidemic of Poliomyelitis is preceded by a carrier epidemic, in which the virulence becomes raised. The carrier, usually an adult, carries the virus for two to three weeks in his naso-pharynx, then it dies out. (2) The incidence of infection during an epidemic, is much greater than the incidence of disease. The presence of the virus has been demonstrated in the naso-pharynx of patients and also of those who give no definite history of having had the disease, and who may or may not have been in known contact with it. It is probable that this latter group harbour the virus in a more or less stable equilibrium with the host, and that traumatic conditions or lowered resistance allow it to ascend the central nervous system. (3) A factor bearing this out, and which has been a common finding with medical men throughout the province where Poliomyelitis has been prevalent this summer, is the abnormally high number of patients complaining of indefinite symptoms such as malais, headache, slight stiffness of neck or back, perhaps an irritated throat, with very little if any disturbance of pulse or temperature. These symptoms are very transitory, and recovery is rapid, but the number of these cases is many times that seen in normal periods, and our belief is that these are mild abortive types of the disease, but are also cases which are capable of causing further spread of the infection. It has also been demonstrated in this district and others, that individuals, whilst they are in the incubatory stage, before any symptoms have appeared, are also

capable of spreading the infection. There is not sufficient reason to believe that the virus of Poliomyelitis is conveyed to man by food stuffs including milk, or insects, or that the disease is associated with insanitary conditions. (1) A survey of the homes where cases occurred showed the following classifications as regards sanitary conditions:—Good 45%, Fair 37%, Poor 18%.

SYMPTOMS

A precise and practical conception of poliomyelitis is obtained only by recognizing the existence of three clinically and pathologically distinct stages in its evolution. The picture which will best convey the progress of the disease is *first*, that of a general infection in a sick child or an indisposed adult; *second*, a meningitic invasion, and *third*, in some cases, an extension into the grey matter of the cord, with weakness or paralysis, or localized nervous symptoms. These stages may sometimes be clinically simultaneous, but usually meningeal signs precede evident paralysis.

Initial Stage or Systemic Symptoms. Recognition of this stage is doubly important for the protection of contacts, and for the institution of measures of treatment. It may last only a few hours and be unrecognized, or it may extend to four or more days. The symptoms may simulate any of the indefinite illnesses of childhood, and in the presence of an epidemic of Poliomyelitis, it is well to treat sick children having a fever without a definite diagnosis, as possible cases of Poliomyelitis. Still, there are groups of symptoms which are very suggestive.

Fever is the most common single symptom and may be of any grade. The average temperature amongst our cases was not above 101 and frequently not more than 99.5, but, and this is important, usually accompanied by a pulse rate out of proportion to the temperature. Very seldom was there a rate below 100 and in some cases it ran as high as 160. Average 110-130.

Headache was the next most common symptom occurring in 43 of the cases. Very often it was severe, but in 50% it was recorded as moderate or slight.

Malaise was noticeable in practically every case. The older age groups especially, complain of feeling tired and very weak previous to the onset of more acute symptoms.

Nausea is perhaps the next most persistent finding, occurring in over 60% of the cases, and usually one of the earliest symptoms to be present.

Vomiting, if it occurs, is not usually prolonged, and by many patients is attributed to indiscretions in the diet and not to the disease.

Sore throat was not a major symptom and was recorded in only eight of the cases, although examination of a majority showed a mild injection of the whole posterior pharynx.

Intestinal symptoms are frequent. There may be abdominal cramps and either diarrhoea or constipation. Of this series, ten had diarrhoea while fourteen had constipation.

One of the common symptoms which frequently aid diagnosis in this stage, is *drowsiness*.

The opposite symptom, that of *restlessness* or irritability, was also encountered, sometimes in the same patient. In our series we found 50% had drowsiness, while 35% exhibited restlessness, and in six cases both were found in the same patient.

Two other symptoms which are frequent, and which when present tend to confirm the diagnosis, are *sweating* out of proportion to the air temperature, and *retention of urine*. The latter was noted in about one third of the cases.

Herpes is very rare, although there may be morbilliform rashes appear which at times may be mistaken for other of the exanthemata.

It may be argued that there is nothing distinctive about this clinical picture, that the symptoms enumerated are merely those which may occur in any sick child, and which may pass off without a definite diagnosis being made, but the combination of fever, rapid pulse, headache, drowsiness, irritability, especially when combined with flushing of the face, abnormal sweating or retention of urine is enough to make a tentative diagnosis of Poliomyelitis if there are frank cases occurring in the district. (4) The onset of this systemic stage is frequently insidious. We have histories of eight cases where there was a definite prodromal period of several days before definite symptoms appeared, but it must also be remembered that this stage may pass very rapidly into involvement of the C.N.S., so that closest observation is indicated in every case, no matter how slight the symptoms.

From this stage the condition may clear up rapidly, and complete recovery ensue. But it is equally important to remember that there may be a remission period of some hours or even days, when the temperature falls to normal, and the child appears to have recovered. But this is followed by a secondary rise of temperature and an accentuation of the disease. All too tragically was this phase brought out to us during the epidemic, two deaths and one severe residual paralysis resulting from the mistaken idea on the part of the mother or patient, that this period of remission constituted a cure.

Pre-Paralytic Stage.

Next we come to a consideration of the *Pre-paralytic stage* or the *Meningeal symptoms*. This stage indicates subarachnoid or meningeal invasion. It varies in intensity and duration, but on the whole its duration is short, and it ends either in speedy recovery or passes into the paralytic stage within a few hours.

The greater part of the symptoms already enumerated are continued or renewed, and in addition others make their appearance, chief among which are *pain on spinal flexion*. Pain on forward nodding of the head or especially on forward bending of the lower spine is very frequent and

characteristic. This sign was present in 87% of the Morton cases. Kernigs sign may or may not be present, depending on the degree of meningeal involvement.

Hyperaesthesia is also a symptom attributable to meningitis. The tenderness may be of the skin, on deep pressure of the muscles, or on motions of the joints. The hypersensitiveness may be general in one part of the body only and, if so, often indicates the site of future paralysis.

Loss of sensation was found in some cases, but was a minor feature.

Other phenomena attributed to irritative lesions of the nerve cells are *tremor*, brought out especially if the limbs are extended unsupported or if muscular effort is attempted, and *muscular twitching*, this being present in 40% of the cases.

There is also noticeable lack of co-ordination of motion, which may be noticeable in performing simple motions with the hands, or as an unsteadiness in gait or on standing.

Another noticeable feature of this stage, and one equally difficult to describe, is the *general facial appearance* of the patient. He has what might be termed a definite apprehensive or frightened appearance. The eyes are shiny, almost glassy, and the whole appearance is one indicative of fear or alarm.

Examination of the *reflexes* in this stage also yields valuable information. It is likely to find irregular increases in reflex response, with perhaps some spasticity. We have found some disturbance of reflex in practically every case that has reached this stage. There is usually an exaggerated response for some hours before it passes off into diminution or loss. A unilateral increase or decrease is of more significance than any symmetrical change.

An examination of the *spinal fluid* is undoubtedly of value in this stage, in many cases, and will often serve to clear up an otherwise obscure diagnosis. There is increased pressure, with a clear or nearly clear fluid containing no organisms, and with a cell count of over ten, maybe up to 1,000, with increased albumin and globulin. Without a doubt, if meningeal symptoms are at all pronounced, the procedure of spinal puncture should be followed in order to relieve pressure, and rule out other forms of meningitis. But, in the initial stage previously described, before meningeal symptoms have appeared, the fluid is normal. Our experience has shown us that if the diagnosis can be made in this initial stage, before spinal fluid changes have occurred, that the patient's chances for recovery are greatly enhanced, and even in the second stage of meningeal irritation, a thorough examination of the patient and consideration of the history will, in the usual case, enable a diagnosis to be made as positively without as with a lumbar puncture. (4) We feel, therefore, that where clinical symptoms are definitely suspicious, and where it is not convenient or possible to carry out a lumbar puncture under

proper aseptic precautions, or where facilities are not available for prompt examination of the fluid, that treatment should on no account be delayed due to lack of corroborative spinal fluid findings.

The Paralytic Stage, or stage of invasion of the central nervous system.

The paralysis, when it occurs, is typically flaccid. There may be increased tonicity in the early stages but it never lasts. The paralysis comes on as a rule 4 or 5 days after the initial onset of the disease. Certain muscle groups are much more commonly affected than others, the occurrence of foot drop testifying to the frequent involvement of the lower leg muscles. Of the eight cases paralysed in our series, 3 had paralysis of the right leg, 4 of both legs, and two of these with one arm involved, and 1 had paralysis of the right arm alone. Four of the fatal cases developed an ascending type of paralysis—so called Landry's paralysis, which began in the legs about the 4th day, and rapidly spread upwards involving the sphincters of the bladder and bowel, the arms, the diaphragm, the intercostals, and finally death supervened due to paralysis of the throat. A fatal outcome in three other cases was due to paralysis of the bulbar centers of respiration, so called bulbar or cerebral types, causing difficult swallowing, aphonia, and regurgitation through the nose. In all of these cases, cerebral irritation was a marked feature throughout the entire illness.

DIAGNOSIS

It is evident that the diagnosis of poliomyelitis is not a simple matter, depending on a single factor or sign, but that the whole history and physical examination must be taken into consideration, and when that is done, there are enough idiocyncrasies and predilections of the disease to enable a diagnosis to be made with as great certainty as is usual in the diagnosis of other diseases, without what was formerly considered the essential feature of the malady; permanent paralysis.

TREATMENT WITH SERUM

The administration of the so called convalescent serum prepared by Professor F. C. Cadham at the Provincial Bacteriological Laboratory was used in every case of the 47, with only one exception, and an analysis of the results shows some striking comparisons. Thirty of the cases received serum within 30 hours after initial onset, and of these thirty, twenty-eight have made complete recovery without any sign of residual paralysis. These exceptions are both children who were not kept in bed after the initial symptoms subsided, and a very mild partial paralysis of one leg was noticed only after they had been on their feet for some time. Among the 28 cases which have made rapid and uneventful recovery, are many cases of striking results following the use of serum. Many of these cases, although of short duration, were very acutely ill at the time

of treatment, and without exception there was a rapid loss of symptoms in the 24 hours following, and a marked improvement in the general well being of the patient. Many histories could be cited to exemplify the remarkable change which occurs in these very early cases which receive serum. Nearly all will tell the same story and many will name the very hour at which they began to feel better. There is apparently a latent period of from 8 to 14 hours after administration, before beneficial effects are felt, and the rapid response following that, is perhaps the most satisfactory thing from both the patients and physicians standpoint that I have experienced, excepting perhaps, the lancing of a quinsy in the acute stage.

But there is also the other side to the story, when we begin to consider cases in which the administration of serum was delayed. Four cases received serum 50 to 60 hours after onset, and of these, one died of an ascending type of paralysis, one is permanently crippled in both legs, and for two weeks had paralysis of the bladder and one arm as well, one has paralysis of one leg, and only one made recovery. Seven cases were given serum between 3 and 4 days after onset, with the following results; four died of typical respiratory paralysis within seven days, one has both legs paralyzed, and two recovered. Four other cases were given serum in periods from 4 to 6 days after acute onset. Two are dead and the other two have residual paralysis in both legs. If the cases are classified according to stages of infection when they received the serum, we find the following results:—

Twenty-two received serum in the initial or infectious stage, and twenty-one completely recovered and one has slight partial paralysis of one leg.

Nineteen received serum in the second or meningeal stage (pre-paralytic). Ten recovered, two have residual paralysis, and seven died.

Five received serum in the paralytic stage, and of these one died and the other four still have residual paralysis.

The most noticeable feature of these latter cases, and just as noticeable as the response in the earlier cases, was the almost complete lack of response or disappearance of symptoms following the administration of serum. Of the 14 cases which received serum after 36 hours, only three have recovered without paralysis, which, to my mind, is only additional proof of the virulence of the infecting virus in this epidemic. In five cases, repeated doses of the serum were given failing a response to the first, and there was no noticeable diminution of symptoms at all. It is believed that once the virus has combined with living cells it cannot be neutralized by human serum. Thus, serum is too late to preserve susceptible cells, unless given before the virus-cell union occurs (3).

It has come to be my firm conviction that the reason more universal acceptance and favor for

the serum has not been forthcoming in the literature, is that physicians have waited too long before making a diagnosis, and thus have missed the period during which the serum would have such marked effect. We feel that if one waits for definite symptoms of meningeal involvement such as disturbance of reflexes, marked stiffness or rigidity of the neck, positive spinal fluid findings, muscular twitchings, etc., that there is grave danger of missing the opportunity of saving these patients from possible paralysis or death, and that this factor alone is perhaps more than anything else responsible for the high morbidity and mortality rates which have been the experience in poliomyelitis statistics in the past.

PUBLIC HEALTH ASPECT

One word in conclusion with regard to a public health aspect of the disease. As it appears from our experience at least, that to be of value the serum must be given very early, clearly then that is only possible if the doctor can see his patient early. The public has been notified by the press, by pamphlets, by radio, by local physicians, and by public health nurses, of the importance of calling medical aid at the earliest possible moment. In many cases only the matter of the expense involved has caused parents to delay in calling a doctor, with sometimes, disastrous results. The action of many of the municipalities this fall in granting free treatment to all cases and free diagnosis to all suspicious cases, cannot be too highly commended. It has been the experience in nearly every district where poliomyelitis has occurred this summer, that the first cases were not seen by medical men until paralysis had developed. The institution of this system in the Municipality of Morton in August has resulted in not one case having reached the paralytic stage since that time. In all the areas concerned, the public have been notified by the press and by published notices that such services are available and they are taking full advantage of it. Doctors in these areas report that while they are seeing many cases which do not require treatment, they have yet to find a patient in the advanced stages of poliomyelitis in the first consultation. This association might let us consider this a very forward step in the field of preventive medicine, and only by full co-operation of all parties concerned can effective control of this most dreaded of all infectious diseases be maintained.

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Anterior Poliomyelitis—Manitoba, 1936

By

C. R. DONOVAN, M.D. (Man.), D.P.H. (Tor.)
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Manitoba

The first case of epidemic Poliomyelitis reported in Manitoba this year was from Ochre River on May 2nd, 1936, then during the third

and fourth weeks of June three cases occurred in Morton Municipality. During the following week a case was reported from the Municipality of Grey. The week of July 13th brought in a report from Dufferin and another from the Morton area, and then in a few days the cases began to crop up rapidly in Boissevain and Morton, so that by the middle of August twice as many Poliomyelitis cases were reported from this Municipality as in all the rest of the Province. From this time on the cases in the Morton area rapidly fell off, while in the remainder of the Province a definite increase is noted nearly every week until as Table I shows the week of September 14th reports 46 cases, which so far is the high point, and may be the peak.

The cases have largely been confined to the South Central portion of the Province, but the general direction of the extension from Morton was East and North-East. Each week cases were reported from fresh municipalities, and on August 13th, 1936, the first case was reported in Greater Winnipeg.

Up to date Poliomyelitis has been reported from the following municipalities since June 15th, 1936, and just about equals the cases reported during 1928 outside Winnipeg:—

Assiniboia 1, Boissevain 14, Cartier 1, Brandon 4, Dufferin 1, Gretna 3, Grey 2, Langford 1, Louise 3, Lansdowne 2, Kildonan West 2, Manitou 1, Montcalm 1, Morden 2, Morton 30, North Norfolk 1, Neepawa 3, Oakland 2, Pembina 1, Portage la Prairie 3, Riverside 1, Rockwood 2, Roblin Rural 2, Rosedale 2, Selkirk 10, Stanley 8, Strathcona 1, St. Andrews 3, St. Boniface 2, St. James 1, St. Vital 1, Turtle Mountain 3, Unorganized 1, Wawanesa 2, Whitewater 8, Woodworth 8.

TABLE I
ANTERIOR POLIOMYELITIS IN MANITOBA
Reported each week from June 15 to
September 20th, 1936

| Week Beginning | Morton Area | Remainder of Province, less Greater Winnipeg | Greater Winnipeg | Total |
|------------------|-------------|--|------------------|-------|
| June 15..... | 1 | | | 1 |
| 22..... | 2 | | | 2 |
| 29..... | | 1 | | 1 |
| July 6..... | | | | 0 |
| 13..... | 1 | 1 | | 2 |
| 20..... | 7 | | | 7 |
| 27..... | 5 | 5 | | 10 |
| August 3..... | 13 | 2 | | 15 |
| 10..... | 6 | 5 | 2 | 13 |
| 17..... | 6 | 11 | | 17 |
| 24..... | | 10 | 2 | 12 |
| 31..... | 1 | 9 | 4 | 14 |
| September 7..... | 1 | 17 | 10 | 28 |
| 14..... | 1 | 24 | 14 | 39 |
| | 44 | 85 | 32 | 161 |

Immunity Problems of Poliomyelitis

By

F. C. CADHAM, B.A., M.D. (Man.), F.R.C.P. (C.)
*Professor of Bacteriology, University of Manitoba,
Bacteriologist, Department of Health, Manitoba*

Poliomyelitis is a disease of paradoxes. It is called infantile paralysis, but infants are comparatively immune and adults are frequently stricken, especially in this epidemic. Some believe the disease is highly infectious, yet only one child of a group or one person in an isolated community may be attacked. It is a disease of the summer months in temperate climates and comparatively rare in the tropics. It kills or paralyzes in a short space of time or leaves the patient apparently unharmed. The immunologic reactions fail to conform to what we would expect of a virus disease.

The immunology of this disease is of such amazing complexity that it would be impossible and of little value at the moment to discuss these difficult and controversial problems.

The infective agent, a virus estimated to be about 0.2 microns in size, has been isolated and cultured and from the culture the disease has been again transmitted, thus fulfilling Koch's postulate. It has been established that the virus enters through the nasopharynx, travels largely if not exclusively by the neural pathways. Acute poliomyelitis involves the grey matter of the spinal cord, the nuclei of the brain stem and the walls of the third ventricle. The prodromal symptoms indicate an initial systemic invasion.

As a rule sudden in onset and as suddenly over, death, paralysis or recovery. What occurs?—is the virus destroyed in the body by similar processes of immunity that occur in other infectious diseases—by leukocytic activity—there is little evidence—by humoral activity—possibly, but if so, it is obvious that the development of antibody immunity needs must be startlingly rapid. Experience shows that in other infectious diseases time is required for the development of antibodies. These fundamental problems of the disease have been subject to an immense amount of experimental work in an attempt to determine or evolve some sound method of prevention or therapy.

TRANSMISSION

How is the virus transmitted? Peculiarly seasonal in incidence, naturally attention is directed to a possible insect vector, but no insect has been incriminated; in fact, epidemics have occurred in localities and at times that would appear to exclude such a type of vector.

The belief is that it is transmitted by healthy human carriers. The virus has been repeatedly recovered from the nasopharynx of persons in normal health. Can we isolate these carriers? At present by no known practical method. Why do not these people succumb to the disease?

Possibly because they have a general immunity or a local tissue immunity of the nasopharynx, or possibly an anatomical mechanical factor is present that prevents the invasion of the virus.

Practical application of the principle involved in the mechanical blocking of the paths of ingress has recently been attempted by applying to the nasopharynx antiseptics that congeal the secretions—picric acid, tannic acid, alum, mercuriochrome or alcohol.

IMMUNIZATION

At once, since it is a virus disease, we consider the possible active immunization of the population as practiced to control smallpox. Kolmer has prepared and administered an attenuated vaccine of the virus. Brodie has used a killed culture. Twenty thousand persons are said to have received these vaccines. Nine cases with five deaths occurred in this group, and Dr. Leake of the United States Public Health Service points out that although any one of these cases may have been unconnected with the vaccine, the implication of the series as a whole is clear. At present we must hesitate. I believe, however, that our hope in conquering this dread malady lies in the further improvement of this method.

Failing active immunization, the question of passive immunization arises. Up to the present efforts to immunize animals so that a neutralizing antibody of high titre might be obtained have failed; the virus lacks antigenic value for animals. However, this line of investigation also is of promise.

Finally, we must consider the transference of human serum which is known to contain neutralizing antibodies.

That these neutralizing substances appear in the blood following an attack of poliomyelitis has been abundantly demonstrated. They have also been demonstrated in the blood of over 80% of adults who have no recollection of having suffered from an attack of poliomyelitis and to complicate the question further these neutralizing substances have been found present in horse serum, venom serum and placental extracts.

It is conceived that the persons who have not suffered from the disease but whose blood shows the presence of antibodies have been immunized by a non-symptomatic attack, possibly by an attenuated type of the virus. We have here an analogy to diphtheria—the high protective power of the infant, low antibody content of the blood after one year of age, gradually rising in the general population to adult life; however, in poliomyelitis the problem becomes involved, since persons in whose blood neutralizing substances have been present have been known to contract the disease. Moreover, there are 17 cases on record of a second attack.

It is established that the transfer of human serum is the only practical method of therapy aside from prevention that at present is available.

VALUE OF CONVALESCENT SERUM

The efficacy of convalescent serum in poliomyelitis is the subject of controversy. Park of New York scouted the value as the result of a summary of observations made in the New York epidemic of 1931, even though the statistics of that epidemic indicated less paralysis and a lower death rate in the treated cases; later the New York Academy of Medicine pointed out the untreated group of cases recorded for statistical purposes were a much milder group than the treated group.

Brodie of New York stated he was doubtful if beneficial results were obtained by the experimental method in monkeys, but, as has been pointed out, the results observed in the therapeutic tests of the serum on monkeys does not necessarily postulate that the same would hold for the human type of the disease. On the other hand, Schultz and Gibbard using convalescent serum reduced the death rate in monkeys by 23%. It is of interest to note that while monkeys are highly susceptible to the virus not all are so; the spider monkey of South America appears to be resistant.

Abundant evidence exists that benefit follows the administration of convalescent serum. In the California epidemic, the Manitoba epidemic of 1928, the Michigan epidemic, the Chicago cases and in the Australian epidemic favourable results were noted following the use of convalescent serum. Jensen, describing the extensive epidemic in Denmark of 1935, states "There is a direct and positive justification for the use of serum, in that skilled observers independently again and again have made the clinical observation of a prompt improvement of the general condition of the patient following serum therapy. In a number of cases this improvement was both subjectively and objectively so prompt that the influence of serum was almost unquestionable."

In a recent editorial review of this subject in the *Journal of the American Medical Association* this statement is made—"If future epidemic results can duplicate those mentioned, the treatment of acute poliomyelitis will be satisfactory and harmless to the patient. There is no other treatment that is even of debatable value. The early and continued use of orthopedic measures will improve results in the acute paralytic disease and in cases in which only paresis appears," and Harmon in a recent comprehensive review of the subject of poliomyelitis states "Convalescent and other specific serum therapy should be continued as there is no evidence that it is not of value, on the other hand symptomatic improvement following the administration of the serum is almost universal."

During the past twelve years we have prepared convalescent serum in the Provincial Laboratory; my experience over that period leads me to believe that the treatment is decidedly valuable. Over 1,200 patients have been treated by the serum from the Manitoba Provincial Laboratory and the records of results are uniformly favourable.

It is accepted that the serum is valuable only when administered in the preparalytic stage. Physicians frequently tax me with the question "how can you judge results when the serum is given early, probably the patient may never have had poliomyelitis and the statistics are then misleading, only apparently proving the value of the serum." Strange to say, this factor works in exactly the opposite way. Let me explain. In the epidemics in Manitoba twice the number of vials of serum have been administered as there are cases reported. I estimate of the number of cases treated but unreported some 15% to 20% may not have had poliomyelitis but the other 80% or over had poliomyelitis. A number of these patients showed all the clinical symptoms, including high cell count in the spinal fluid. Sick a day, serum, recovery the next day. Now if the records are taken from the reported cases—and they have been—what then of the other 80% of recovered cases?

I do not believe the serum treatment even in the preparalytic stage is infallible. Far from it. Neither is anti-diphtheritis serum infallible. There probably exists a fulminating type of the infection in which no form of therapy is of avail; then, too, who can determine at what moments the nerve tissue may have been damaged past repair. The element of time appears to be the important factor; early diagnosis, early treatment. The sudden onset and sudden result demonstrate all too clearly the rapidity of the development of immunity, or its failure. In the abrupt battle, if we stimulate the defensive mechanism of the body even slightly, it may be the deciding factor for a fortunate outcome. I am not convinced that the benefit obtained by the use of the convalescent serum depends entirely upon the passive transference of virus neutralizing substances.

ADMINISTRATION OF SERUM

A constant demand arises for the serum for prophylactic purposes. A two weeks' immunity might be so transferred and even that is questionable. Then, too, some physicians do not understand why we cannot keep all supplied with serum to hold in reserve.

Every patient in Manitoba so far as we know in the past 12 years has been able to obtain the serum when necessary and we will endeavour to keep up that record, but I trust that the profession will not take it amiss if I remind them that the serum is not readily come by and it is difficult and expensive to prepare.

I prepare the serum by a different method than is adopted in other centres and have advised the intramuscular route of administration. This advice was based on my experience with the experimental production of antibodies in animals. Howitt of the University of California has since confirmed experimentally the efficiency of this method of administration of convalescent serum in contra-distinction to the intravenous or intrathecal route.

We distribute a pooled serum in vials. The serum is obtained from the blood of selected donors. A Wassermann test is made on each blood. I recommend the administration of the entire contents of the vial. As a rule physicians have limited the amount given to infants. A 20 cc. all glass syringe is required. We also request that once a vial is opened or partly used that the remainder of the contents be discarded.

Experience at the Winnipeg Hospitals for Infectious Diseases

Dougald McIntyre, M.D., Assistant Superintendent of the Winnipeg Hospitals for Infectious Diseases, discussed their experiences with this disease. He stated that they had numerous cases admitted for observation with a provisional diagnosis of poliomyelitis. These were all investigated carefully and a certain proportion were found to be negative and were discharged; all such cases were kept under observation for at least nine days.

The experience with the lumbar puncture had been variable, some had a comparatively low cell count of 20 to 30, and some others had a count as high as 420.

With regard to the clinical aspects of the disease, he stated he had nothing to add to the features which had already been emphasized by the previous speakers.

As a result of their experience he is of the opinion that if the serum is given early, that is, within twelve to twenty-four hours of the onset of symptoms, that the chances of avoiding paralysis are very good. He considers the serum of definite assistance in the treatment of the disease and in fact the only therapeutic agent which is of tangible value in this condition.

Dr. McIntyre suggested that it is very difficult to accurately assess the value of serum therapy and in attempting to do so it must be kept in mind that poliomyelitis like other diseases varies in severity and in its clinical manifestations in different epidemics.

He suggested that the possibility of giving much larger doses of serum might be considered. It is possible he contended that larger doses might be of value in the more serious cases, or in the cases which were seen some considerable time after the onset. Their experiences with other forms of serum therapy, for example in diphtheria, lead him to suppose it might be of value in some cases of poliomyelitis to give much larger doses. He found nothing to intimate that there was any special risk associated with the giving of the serum.

GENERAL DISCUSSION

Gordon Chown, F.R.C.P. (C.), Senior Physician to the Winnipeg Children's Hospital, discussed the question of the increase in cell count in the spinal fluid, which was necessary to confirm the diagnosis of poliomyelitis. He stated he had seen two cases with clinical symptoms which, in his opinion, justified the provisional diagnosis of poliomyelitis and in which the cell count in the spinal fluid after admission to the hospital was 50 cells in one case and 20 cells in the second case. The two cases were brothers and were admitted to hospital September 5th and 7th respectively. These cases had been given the usual dose of serum and in each instance had been classified as suspect poliomyelitis. He was of the opinion that these should be classified as anterior poliomyelitis and recorded as such. He asked Professor Wm. Boyd, the Pathologist, his opinion as to what constituted an abnormal cell count in the spinal fluid of children.

Wm. Boyd, F.R.C.P. (Lond.), Professor of Pathology, University of Manitoba, replying to Dr. Gordon Chown's question, suggested that an increase of cells beyond 10 in a spinal fluid count was abnormal.

F. A. Benner, M.D., stated that it had been intimated there might be some danger in the doing of lumbar punctures. He was of the opinion as a result of his experience that there was no special danger in doing lumbar punctures for diagnosis, and pointed out that in meningitis they were done frequently in some cases, and also intra-theal injections of serum given. He asked if there was any special danger in these cases of which he might not be aware.

Replying to Dr. Benner's question, Dougald McIntyre, M.D., Assistant Superintendent of the King George Isolation Hospital, stated that he did not believe there was any special danger in the doing of lumbar punctures in suspected cases of poliomyelitis, and certainly one was justified in including this investigation in a suspected case during the course of the epidemic. He emphasized, however, that the practitioner should not feel that a diagnosis without a lumbar puncture was impossible. He added that he had seen cases in which the cell count on lumbar puncture had been normal and then, a day later, the cell count in the spinal fluid was definitely increased—in one case it was 80.

Replying to Dr. Gordon Chown, Dr. McIntyre stated that he was of the opinion that there were a great many mild cases with a slight increase in cells in the spinal fluid for example, 20 to 30, which should be classified as poliomyelitis. The diagnosis referred to by Dr. Chown in the two cases was the provisional diagnosis on admission to hospital.

M. S. Lougheed, M.D. (Man.), B.Sc. (Oxon.), Bacteriologist Department of Health, discussed the incidence of the disease in the City of Winnipeg, and pointed out that the greatest increase in the number of cases had occurred within the last few days.

W. W. Musgrove, M.D., asked Professor Cadham to express his opinion as to the value of whole blood in poliomyelitis and the method he would advise in giving it.

Answering Dr. Musgrove's question, Professor Cadham stated that he was of the opinion that if no serum was available that adult whole blood 25 c.c.s. by subcutaneous injections might be of value.

A. A. Murray, F.R.C.S. (C.), discussed the question from the point of view of the orthopedic surgeon. He was interested in the treatment of cases which had developed paralysis. He pointed out that it was essential for the practitioner in attendance to see such cases frequently during the weeks and months immediately following the acute illness. He also pointed out that a group of muscles which might be the weakest in a limb at a particular time should be put at rest by suitable splinting, but that in a comparatively short time another and opposing group of muscles might become relatively weakened. It would then be necessary to adjust the splintage in order to deal with the new situation. He was of the opinion that there were now a sufficient number of cases to warrant that some definite programme should be initiated by the Department of Health looking towards the future care of the indigent patients who are left with permanent partial paralysis. He suggested that it is imperative that this action be taken now because it is much easier, better for the patient and more economical to prevent deformities rather than to correct them.

The question was raised as to the efficiency of the Picric Acid nasal spray as prophylactic against the epidemic of anterior poliomyelitis. Replying to the question of efficiency of the spray, Dr. M. R. Elliott stated that it had been recommended by various medical men who had experience of epidemics in other places. In their own experience it seemed to help prevent the spread of the disease. In one area in which it had been introduced there had been no new cases among children who had been given the full course of prophylactic treatment. There had been two cases where the spray had been used but in both cases the spray had been used only four days and it was presumed that these children had been infected before the use of the spray was started. Dr. Elliott stated that it was too early to form a definite opinion as to the value of the spray but there was sufficient justification as a result of the experience of others and the experience with the present epidemic to recommend its use as prophylactic agent.

F. W. Jackson, D.P.H. (Tor.), Deputy Minister of Health, addressed the meeting briefly and thanked the Winnipeg Medical Society for arranging a symposium on Anterior Poliomyelitis, and suggested that it would be of great value to the medical practitioners and to the Department of Health. He stated that, in his opinion, it was clear, as a result of the experience of his Department with this disease, and as a result of the discussion which had taken place, that control of the disease depended upon early diagnosis and early administration of convalescent serum.

WHAT EVERY WOMAN DOESN'T KNOW— HOW TO GIVE COD LIVER OIL

Some authorities recommend that cod liver oil be given in the morning and at bedtime when the stomach is empty, while others prefer to give it after meals in order not to retard gastric secretion. If the mother will place the very young baby on her lap and hold the child's mouth open by gently pressing the cheeks together between her thumb and fingers while she administers the oil, all of it will be taken. The infant soon becomes accustomed to taking the oil without having its mouth open. It is most important that the mother administer the oil in a matter-of-fact manner, without apology or expression of sympathy.

If given cold, cod liver oil has little taste, for the cold tends to paralyze momentarily the gustatory nerves. As any "taste" is largely a metallic one from the silver or silverplated spoon (particularly if the plating is worn), a glass spoon has an advantage.

On account of its higher potency in Vitamins A and D, Mead's Cod Liver Oil Fortified With Percomorph Liver Oil may be given in one-third the ordinary cod liver oil dosage, and is particularly desirable in cases of fat intolerance.

—Advt.

ANAHAEMIN B.D.H.

The British Drug Houses Limited report that they have undertaken the manufacture on a commercial scale of the active haematopoietic liver principle described by Dakin and West (Journ. Biol. Chem. 1935, cix, 489).

The clinical trials of Anahaemin, B.D.H. arranged by the Medical Research Council, London, England, were published in the "Lancet," February 15th, 1936, page 349. These trials demonstrate that by the use of Anahaemin, pernicious anaemia can be treated successfully with the minimum of inconvenience and expense.

One injection of 2 c.c. of a solution containing 200 mgm. of Anahaemin, produces an immediate reticulocyte response, followed by a striking increase in the number of red blood corpuscles which is sometimes maintained for a period of over thirty days. In many cases, one such injection at monthly intervals will constitute effective treatment, indeed, according to the report "... no other liver extract given in the small amounts used ... has produced such striking results."

Anahaemin, B.D.H. is available in ampoules of 1 c.c. and 2 c.c. in boxes of 3's and 6's, from principle drug stores, and descriptive literature is available on application to The British Drug Houses (Canada) Limited, Terminal Warehouse, Toronto.

—Advt.

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EDITORIAL

The Epidemic of Anterior Poliomyelitis

For several weeks now there has been an outbreak of Anterior Poliomyelitis in Manitoba of epidemic proportions. In view of this situation the Winnipeg Medical Society gave over their first meeting of the season on September 18th to a symposium on Anterior Poliomyelitis; the papers and the resumé of the subsequent discussion are published in the clinical section.

This meeting had the largest attendance of any of the Winnipeg Medical Society meetings for some time. Those who had special experience with the epidemic of 1928 discussed the features of the disease at that time, and the medical men who are engaged in combatting the present epidemic described their experience with the disease in the past few weeks. Most of the discussion dealt with the control of the disease during this acute stage, but A. A. Murray, F.R.C.S. (C.), raised the question of making provision of indigent cases with residual paralysis. It was understood that the Department of Health are initiating a programme for the care of these cases as the necessity arises.

F. W. Jackson, D.P.H. (Tor.), Deputy Minister of Health of Manitoba, congratulated the Executive of the Winnipeg Medical Society for arrang-

ing this excellent symposium on poliomyelitis. It remains, however, to record what everyone at the meeting must have felt, and that is the appreciation of the prompt and energetic efforts made by the medical officers of the Department of Health of Manitoba during the present epidemic. Every general practitioner who has come into daily contact with the necessity of diagnosing and treating anterior poliomyelitis must be grateful for the assistance which was given to him by the officers of this Department.

It is apparent that the opinions expressed by those who read papers are probably only tentative; they may be confirmed or modified, particularly the value of convalescent serum, when all the statistics are available at a later date.

—C. W. MacC.

Minutes of Executive Meeting

Minutes of a Meeting of the Retiring Executive and the Executive Elect of the Manitoba Medical Association, held in the Medical Arts Club Rooms on Friday, September 18th, 1936, at 6.30 p.m.

Present.

| | |
|----------------------|-------------------------|
| Dr. F. G. McGuinness | Dr. C. W. Wiebe |
| Dr. Geo. Clingan | Dr. W. E. R. Coad |
| Dr. F. W. Jackson | Dr. C. W. Burns |
| Dr. E. S. Moorhead | Dr. S. Bardal |
| Dr. F. A. Benner | Dr. J. F. Wood |
| Dr. O. C. Trainor | Dr. P. H. T. Thorlakson |
| Dr. S. G. Herbert | Dr. E. K. Cunningham |
| Dr. F. D. McKenty | Dr. W. S. Peters |
| Dr. R. E. Dicks | |

Following dinner the Chairman, Dr. F. G. McGuinness, called the meeting to order and requested that the minutes of the last Executive Meeting held on May 13th be read by the Secretary.

It was moved by Dr. F. D. McKenty, seconded by Dr. W. E. R. Coad: That these minutes, having been published in the Review, be taken as read. —Carried.

Dr. F. G. McGuinness, Retiring President, expressed appreciation to the members of the Executive for their support during the past year, and turned the business of the meeting over to the incoming President, Dr. Geo. Clingan, who replied to Dr. McGuinness' remarks.

Dr. Clingan advised that Dr. Moorhead wished to address the meeting.

Dr. Moorhead reported on certain negotiations he had with the Unemployment Relief Department re. refractions; copy of the report is on file. He also advised that, owing to the great demand for statistics of the medical relief scheme, it would be necessary, in his opinion, to employ an extra stenographer in order that the statistics might be of the greatest possible value.

Appointment of Committee on Federation.

Dr. Jackson read resolution passed at the meeting of the Canadian Medical Association Council in Victoria, and advised that Dr. Routley would be passing through Winnipeg next Friday, and it was essential that the Committee be appointed now.

Following discussion, it was moved by Dr. F. G. McGuinness, seconded by Dr. F. A. Benner: That Dr. F. D. McKenty be Chairman of Committee on Federation with power to add, and pick his own committee. —Carried.

Dr. Moorhead stated that some consideration should be given to this before October 29th, so that this could be presented to the Executive Committee of the Canadian Medical Association at its meeting by our representative on the Canadian Medical Association Executive.

Date and Place of Annual Meeting.

It was decided unanimously that the next Annual Meeting of the Association should be held in the spring, the date of the meeting to be carefully considered and brought up at the next meeting of the Executive.

Letter from Dr. C. M. Strong.

The Secretary read a communication from Dr. Strong, under date of July 11th, in connection with the right of workmen to choose their own doctor instead of being intimidated to going to Company doctors. Dr. Strong's letter was discussed and it was decided that this matter be left with the Winnipeg members of the Executive to deal with.

Dr. James McKenty's Report on "The Relation of the Profession with Hospitals.

The Secretary read a letter from Dr. A. W. S. Hay, Secretary of the Winnipeg Medical Society, under date of March 23rd, attaching Dr. McKenty's paper on the above subject.

It was stated that this was previously held in abeyance, as it was thought the time was not suitable for action on it. Since this, however, Dr. McKenty has had this article published in full in the "Canadian Doctor." Following discussion, it was decided that a letter should be written to the Winnipeg Medical Society, asking for details as to the disposition of this matter.

Letter from Dr. J. W. Cairns.

The Secretary read communication from Dr. Cairns of Pipestone under date of September 2nd, advising that the Federal Government had taken over the whole relief in that district and the set-up did not include medical services. Dr. Jackson explained the situation and advised that information has been gathered as to what the cost would likely be, as this affects eight municipalities. It was suggested that this matter be brought to the attention of Dr. T. C. Routley on his way through Winnipeg this week, and ask him to keep in close touch with the matter at Ottawa and make sure that provision is made for medical attendance.

It was moved by Dr. J. F. Wood, seconded by Dr. S. G. Herbert: That this be left to the Secretary to take care of and reported back to the next meeting of the Executive. —Carried.

Letter from Medical Council of Canada Re. Specialists.

The Secretary read a communication from The Medical Council of Canada and resolution passed by that body on the control of Specialists in Canada. As the members of the Executive were not familiar with this matter, it was suggested that copies of the report of this Committee and information on the subject be prepared and forwarded to each member of the Executive, and that the matter be brought up at a future meeting.

Letter from Department of Health and Public Welfare: Re. Training Schools for Nurses.

The Secretary read a communication dated September 17th from the Department of Health and Public Welfare, regarding training schools for nurses, stating that the University of Manitoba requested that they be free from any responsibility of designating what hospitals should be allowed to operate training schools. The Registered Nurses Association were making a survey and the Department of Health and Public Welfare wished the opinion of the medical profession.

Following discussion, it was decided that a questionnaire should be prepared and brought for consideration at next executive meeting.

There being a meeting of the Winnipeg Medical Society held this evening at 8.30 p.m., the meeting adjourned and a further meeting of this Executive to be called in one month's time.

University of Manitoba

FACULTY OF MEDICINE

POST-GRADUATE COURSE 1936-37: CLINICAL ENDOCRINOLOGY

- Nov. 18th—Professor A. T. Cameron—Thyroid (General).
- Nov. 25th—H. D. Kitchen, M.D. (Man.)—Non-myxœdematous sub-thyroid states and medical treatment of hyperthyroid states.
- Nov. 25th—G. S. Fahrni, F.R.C.S. (C.)—Surgical treatment of hyperthyroidism.
- Dec. 2nd—Professor A. T. Cameron—Parathyroid, Thymus, Pineal (General).
- Dec. 9th—Bruce Chown, B.A., M.D. (Man.)—Recognition and medical treatment of sub- and hyperparathyroid states.
- Dec. 9th—A. C. Abbott, F.R.C.S. (Edin.)—Treatment of hyperparathyroid states including those associated with kidney lesions.
- Jan. 13th—Professor A. T. Cameron—Pancreas (General).
- Jan. 20th—Professor C. R. Gilmour—Hypoglycaemia.
- Jan. 20th—A. Hollenberg, B.Sc., M.D. (Man.)—Treatment of Diabetes.
- Jan. 27th—Professor A. T. Cameron—Reproduction (General).
- Feb. 3rd—Time reserved for guest speaker.
- Feb. 3rd—J. D. McQueen, F.R.C.S. (C.)—Treatment of disturbances of the menstrual cycle.
- Feb. 10th—Professor A. T. Cameron—The Adrenal Glands (General).
- Feb. 17th—J. D. Adamson, M.R.C.P. (Edin.)—Addison's Disease.
- Feb. 17th—Professor J. A. Gunn—Paroxysmal hypertension.
- Feb. 24th—Professor A. T. Cameron—The pituitary gland (General).
- Mar. 3rd—Lennox G. Bell, M.R.C.P. (Lond.)—Cushing's syndrome, acromegaly, dwarfism.
- Mar. 3rd—O. S. Waugh, F.R.C.S. (C.)—Surgical treatment of pituitary conditions.
- Mar. 3rd—John Hillsman, Ch.M. (Man.)—The pituitary and water regulations.
- Mar. 10th—Professor A. T. Cameron—Anti-hormones and pituitary inter-relationships.

ASCORBIC ACID, B.D.H.

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—Advt.

Department of Health and Public Welfare

NEWS ITEMS

The following is an article written on "Intestinal Intoxication—its Prevention," by Samuel Karelitz. This appeared in the August, 1936, copy of "Preventive Medicine."

"In recent years it has become apparent that the severe symptoms which make up the picture of intestinal intoxication are the result of a disturbance in the chemical equilibrium in the body brought about by the loss of water, salts and organic substances, primarily by way of the gastrointestinal tract, and that the duration and severity of the diarrhea determine the appearance of the clinical picture of intoxication rather than the underlying cause of the initial gastrointestinal upset. Expressed differently, any condition which leads to diarrhea in infancy if permitted to continue will ultimately bring about the state of anhydremia (desiccation or severe water loss) with its resulting blood concentration, acidosis, toxemia and circulatory collapse.

The above facts being the generally accepted views concerning intestinal intoxication, a paper dealing with the preventive aspects of the symptom complex should, therefore, deal with the causes, the prophylaxis, and the treatment of the ordinary gastrointestinal upsets of infancy, especially diarrhea.

CAUSES OF DIARRHEA

The classification of the causes of gastrointestinal disturbances in infancy made by Czerny serves well in the discussion of the subject. He divided the causes of gastrointestinal disturbances of infancy into three groups; those associated with infection, alimentation, and those associated with constitutional peculiarities.

Under the subtitle infection he referred to the harmful effect of enteral infection, such as dysentery; the parenteral infections such as acute upper respiratory infections, otitis media, and pneumonia, and to the injurious substances introduced with food, which had been caused by the bacterial action upon it, or by the harmful products of fermentation and putrefaction brought about by the action of bacteria upon foods within the intestinal tract.

Under alimentary causes he referred to overfeeding, underfeeding, or partial starvation leading to malnutrition, or the feeding of poorly balanced foods, such as those unusually high in the fat or carbohydrate content, or the giving of food difficult for an infant to digest.

Under the title of constitutional predispositions to gastrointestinal disturbances he referred to the cases of inherent intolerance for carbohydrates or fats in quantities usually well tolerated by normal infants, to the hypertonic and pylorospastic infants, and to those now known as allergic.

The causes mentioned, and others which fall under Czerny's classification include most of the causes of gastrointestinal upsets in infancy. I have seen cases of diarrhea illustrating each of these etiological factors which have gone on to the development of intestinal intoxication.

Other causes of diarrhea which have resulted in intestinal intoxication which have come to my attention are the following: a single dose of castor oil, and in another instance a large dose of milk of magnesia given for constipation, brought about diarrhea; sudden weaning of a baby, especially in the summer, even though pasteurized milk was used; an ounce of orange juice given for the first time—the addition of a fairly large quantity of a new food, such as vegetable; general anaesthetic; mechanical injury to the intestine, such as operation, and quite commonly

summer heat, especially when associated with great humidity, or with an abrupt drop in barometric pressure. It is often difficult to determine the exact etiology of a case of diarrhea, for several factors may interplay in bringing about the disturbance.

MECHANISM OF DIARRHEA

In brief, except for the direct action of enteral infection upon the intestinal tract, and the increased activity brought about by nervous stimulation as in the hypertonic or allergic child, diarrhea is produced by either bacterial irritation or by mechanical irritants to the gastrointestinal tract.

Infants in general, and artificially fed infants especially, are more likely to develop diarrhea than are older individuals. This may be due to a number of factors. The gastrointestinal secretions of infants contain less hydrochloric acid and less of the proteolytic enzymes than those of adults. The gastric juice of infants is well able to digest the small fine curds of breast milk, but is not always adequate for complete digestion of the coarse, tough cow's milk curd. The acidity of the infant's gastric juice is ordinarily adequate to prevent the growth of bacteria in the stomach as well as in the upper part of the intestinal tract. Under abnormal conditions, such as parenteral infection, long standing malnutrition (even in breast-fed infants), or abnormal climatic conditions (high temperature and humidity, and sudden drops in barometric pressure) there is a diminution in the gastric acidity. Associated with this diminished gastric acidity the colon bacillus, which is normally found as a harmless inhabitant of the lower bowel, is found in fairly large numbers in the duodenum, and even in the stomach, and by its action on foods and perhaps directly on the intestinal wall, is believed to bring about diarrhea.

Direct mechanical irritation due to foods which the particular infant is not able to digest properly, for example, large, tough milk curds, highly concentrated sugar solutions, the lower fatty acids, coarse vegetable particles, and toxic products of fermentation and putrefaction, may bring about diarrhea.

PROPHYLAXIS OF DIARRHEA

Despite the fact that diarrhea in most instances is a preventable condition, diarrheal conditions in infancy are still important contributing factors, directly or indirectly, to infant mortality. Probably the best prophylactic measure is breast feeding, since human milk is digestible in nearly all infants. By breast feeding the introduction of harmful bacteria is avoided and malnutrition, one of the greatest predisposing factors to diarrhea, and therefore to intestinal intoxication, is less likely.

Pasteurization and boiling of milk are the most important of the prophylactic measures in the prevention of diarrhea in artificially fed infants. Boiling is especially helpful, for in addition to the destruction of bacteria, the milk is rendered more digestible. Boiling of pasteurized milk for one or two minutes is an important safeguard against possible contamination of the milk after it has been bottled.

Evaporated milk can serve as an excellent substitute for pasteurized milk, and has the added advantages of being less costly for those who are unable to afford grade A milk, of being more digestible for premature infants, and less disturbing in many cases of milk allergy. Because of the greater digestibility of evaporated milk it can be used in greater concentration for premature and feeble infants, thus avoiding the possibility of malnutrition. Powdered milks are useful in that they are more digestible and are easily portable during travel.

An adequate daily water intake must be guaranteed. The infant requires a minimum daily fluid intake of approximately 10 per cent of its body weight. Fifteen per cent is optimum under ordinary conditions, and 20 per cent is maximum, unless the weather is unusually hot and conducive to very great water loss by sweating. The infant usually gets its essential fluid requirement in its food. If, however, there is excessive water loss, either as a result of increased body temperature, or as a result of an increased external temperature, extra water must be offered between feedings to avoid dehydration. In like manner adjustment of clothing and room temperature and air circulation is necessary to reduce excessive water loss in the summer.

The use of correct milk modifiers has also been an important factor in the reduction of diarrhea. Dilution of milk with water aids in reducing the size of the milk curd but unless done intelligently is associated with the danger of underfeeding. Acidification of milk by the use of lactic acid is helpful in that it reduces the size of the curd and by increasing the gastric acidity aids in the prevention of bacterial proliferation in the stomach and in the duodenum. In some parts of the United States a formula of acidified evaporated milk, water and karo syrup has become very popular and the results obtained through its use have been quite satisfactory.

The choice of a carbohydrate modifier is under ordinary conditions important only in that it should not exceed 10 per cent of the total volume of the formula. A combination of a simple sugar and a starch totalling 6-7 per cent of the total volume of the formula is most often successful. If the carbohydrate used in the formula seems to be inadequate a change to a different one, preferably one like dextrimaltose, usually suffices.

Finally the factor which most often initiates diarrhea, namely, parenteral infection, must be dealt with promptly and adequately. For example, in the case of acute otitis media it may be wiser to incise the ear drum in the presence of high temperature, severe earache, restlessness, anorexia and vomiting even though the usual indications for paracentesis have not fully developed.

TREATMENT OF DIARRHEA

Since the development of intestinal intoxication depends on the outcome of the treatment of a case of diarrhea, and since diarrhea will occur inevitably despite all efforts to prevent it, this phase of the subject is extremely important. The reduction in the number of cases of intestinal intoxication in the practice of most physicians would indicate that the treatment of simple diarrhea is fairly well and generally understood. Intestinal intoxication still does occur and is associated with a high mortality.

Certain principles of treatment apply to all forms of diarrhea. These are the early recognition and prompt treatment of parenteral infections; rest of the gastrointestinal tract; restoration and maintenance of a fluid and mineral balance; the giving of food adapted to the limited digestive capacity.

Diarrhea in the breast-fed is usually mild, and is treated by merely reducing the period and frequency of nursing until remission takes place. In the partially weaned baby it is best to eliminate the supplemental feedings entirely, and only gradually replace them when the diarrhea is improved.

In artificially fed infants the treatment of diarrhea is more complicated. In the very mild case it may be adequate to eliminate fruits, vegetables, and other accessories such as cod liver oil until improvement is attained. In those more severe, treatment must be more drastic. First the cause should be determined if possible, and if infection is present it should be promptly and specifically treated as is indicated.

In any case of diarrhea the rate of evacuation of the intestinal content is increased and in consequence digestion and absorption are impaired. Under these conditions, giving the usual diet can only do harm by increasing the already existing irritation of the gastrointestinal tract. It is necessary therefore to diminish food in the very mild cases and in the more severe to withhold food entirely, (for periods varying with the intensity of the condition) from one to two feedings in the relatively mild, to 24 hours or longer in the more severe cases. Irritation of the rectum by the insertion of thermometers or rectal tubes should be avoided. Cathartics should be avoided except for those relatively infrequent cases of diarrhea seen early and known to be due to a food irritant, in which cases a single small dose of castor oil or an enema may be helpful. Although this paper deals with a condition occurring in infancy, the rare case of appendicitis should be kept in mind in giving any cathartic.

During the period of starvation fluid balance should be maintained. Water as such or 5 per cent barley or rice flour solutions, or weakened tea, plain or with the addition of 5 per cent added glucose may be administered by mouth. If vomiting or severe abdominal distention are present the fluid requirement may be satisfied by the subcutaneous and intravenous injection of either Ringer's or Hartmann's or physiologic sodium chloride solution. For intravenous injection 5 to 10 per cent added glucose is helpful. If it is necessary to maintain the intravenous injection over a long period a rate of approximately 130-150 c.c. per kilogram of body weight per 24 hours is usually adequate.

When vomiting has ceased and diarrhea has improved feeding is resumed and the quantities and type of food should depend on the severity of the condition. Fat is least tolerated under these conditions. The choice of formula should therefore be made with that in mind. Sour milk, such as buttermilk or protein milk are generally useful, to be replaced by sweet milk mixtures when the diarrhea improves. It is perfectly possible to resume feeding with sweet milk mixtures provided the quantity offered is very small and the daily increases are likewise small. Thus, in the severe cases 10 c.c. of a 2/3 milk, 1/3 water plus 5 per cent added sugar every 2 hours and increased by an equal amount daily has been found to be generally successful. In relatively few instances was protein milk or buttermilk found to be better than sweet milk mixtures when administered as directed above.

In the course of treatment of a case of diarrhea if symptoms such as loss of tissue turgor, fever, loss of lustre of the eyes and continued or increased diarrhea occurs, intestinal intoxication is present. In that case starvation for at least 36 hours, continuous intravenous injections of the fluids mentioned is instituted immediately and maintained until detoxification is complete and until the child is able to take an adequate amount of fluid by mouth. Blood transfusion is regularly given soon after the intravenous drip has been started. We have found it wise not to inject blood if the hemoglobin content is greater than 80 per cent, or until it falls below that level. Feeding in the manner previously referred to for severe cases of diarrhea, is started only after detoxification is complete, abdominal distention, if present, is relieved, vomiting has ceased, and the diarrhea improved.

I have attempted to show that it is possible in one's general practice to avoid to a large extent the occurrence of diarrhea, to treat it adequately when it occurs, and how to cope with the very severe cases which resist the ordinary treatment and become toxic.

COMMUNICABLE DISEASES REPORTED
Urban and Rural - August, 1936.

Occurring in the Municipalities of:

Scarlet Fever: Total 121—Winnipeg 84, St. Vital 8, Argyle 4, Winnipeg Beach 4, Ste. Anne 3, Dauphin Town 2, Gilbert Plains Rural 2, Portage Rural 2, Brandon 1, Fort Garry 1, Hanover 1, La Broquerie 1, Oakland 1, Portage City 1, St. Andrews 1, St. Boniface 1, St. James 1, St. Paul East 1, Tache 1, Transcona 1.

Measles: Total 83—Virden 17, Unorganized 14, Wallace 11, Winnipeg 10, Eriksdale 6, Thompson 4, Oak Lake 3, Bifrost 2, Brokenhead 2, Minto 2, Archie 1, Elkhorn 1, Emerson 1, Flin Flon 1, Harrison 1, Pipestone 1, Portage City 1, Sifton 1, Springfield 1 (Late Reported: May, St. Clements 1, June, Portage Rural 1, St. Andrews 1).

Anterior Poliomyelitis: Total 62—Morton 17, Boissevain 12, Whitewater 6, Stanley 4, Winnipeg 3, Greta 2, Morden 2, Wawanesa 2, Brandon 1, Cartier 1, Kildonan West 1, Killarney 1, Louise 1, Manitou 1, Norfolk North 1, Pembina 1, Portage Rural 1, Riverside 1, Rockwood 1, Selkirk 1, Turtle Mountain 1 (Late Reported: July, Montcalm 1).

Tuberculosis: Total 43—Winnipeg 13, Unorganized 3, Brandon 2, Dauphin Rural 2, Armstrong 1, Birtle Rural 1, Blanshard 1, Dauphin Town 1, Eriksdale 1, Ethelbert 1, Flin Flon 1, Gilbert Plains Rural 1, Grandview Town 1, Hanover 1, Minitonas 1, Portage Rural 1, Rhineland 1, Rockwood 1, Rossburn Town 1, Selkirk 1, Siglunes 1, Springfield 1, Stonewall 1, St. Boniface 1, St. James 1, Tache 1, The Pas 1.

Whooping Cough: Total 28—Winnipeg 13, St. Paul East 7, Eriksdale 3, Franklin 3, Killarney 1, Unorganized 1.

Chicken Pox: Total 22—Winnipeg 15, St. Clements 3, St. Boniface 2, Blanshard 1, Victoria Beach 1.

Diphtheria: Total 12—Winnipeg 6, Rivers 2, Winnipeg Beach 2, Portage City 1, Whitewater 1.

Mumps: Total 7—Winnipeg 4, Springfield 1, St. James 1 (Late Reported: June, Morden 1).

Typhoid Fever: Total 6—Argyle 2, Hanover 2, Portage City 1, Winnipeg 1.

German Measles: Total 5—Flin Flon 1, Roland 1, St. Andrews 1, The Pas 1 (Late Reported: June, Clanwilliam 1).

Erysipelas: Total 5—St. Clements 2, Unorganized 1, Woodlands 1, Winnipeg 1.

Influenza: Total 5—(Late Reported: May, Unorganized 1, June, Morden 1, Portage Rural 1, Unorganized 2).

Lethargic Encephalitis: Total 1—(Late Reported: May, Ethelbert 1).

Puerperal Fever: Total 1—Harrison 1.

Trachoma: Total 1—Winnipeg 1.

Amoebic Dysentery: Total 1—(Late Reported: June, Arthur 1).

Undulant Fever: Total 1—Winnipeg 1.

Diphtheria Carriers: Total 1—Winnipeg 1.

Venereal Disease: Total 133—Gonorrhoea 95, Syphilis 38.

DEATHS FROM ALL CAUSES IN MANITOBA
For the Month of July, 1936.

URBAN—Cancer 46, Pneumonia 13, Tuberculosis 5, Syphilis 2, Influenza 1, Puerperal Septicaemia 1, Typhoid Fever 1, all other causes 203, Stillbirths 9. Total 281.

RURAL—Pneumonia 19, Cancer 16, Tuberculosis 13, Measles 3, Infantile Paralysis 2, Influenza 2, Lethargic Encephalitis 1, Puerperal Septicaemia 1, Whooping Cough 1, Erysipelas 1, Syphilis 1, all others under 1 year 6, all other causes 168, Stillbirths 8. Total 242.

INDIAN—Tuberculosis 10, Measles 2, Pneumonia 1, all others under 1 year 2, all other causes 8. Total 23.

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"The Practitioner"—July, 1936.

This issue contains a symposium on Early Mental Disorders.

"Endocrines and Mental Disorders"—by Sir W. Langdon-Brown, M.A., M.D., F.R.C.P., St. Bartholomew's Hospital.

"Classification and Etiology of the Neuroses"—by Hugh Crichton Miller, M.A., M.D., M.R.C.P.

"Early Symptoms of Mental Disorders"—by R. D. Gillespie, M.D., F.R.C.P., Guy's Hospital.

"The Physical Factor in Mental Disorder"—by J. G. Porter Phillips, M.D., F.R.C.P., London.

"Depression"—by W. R. Reynell, M.A., M.D., F.R.C.P.

"Dementia Praecox"—by Daniel F. Rambant, M.A., M.D.

"Drug Addiction and Alcoholism"—by Sir James Purves-Stewart, C.M.G.C., M.D., F.R.C.P.

"Sexual Perversions"—by J. R. Rees, M.D., London.

"The Practitioner"—August, 1936.

This issue contains a symposium on Venereal Disease.

"Some late Effects of Venereal Disease"—by L. W. Harrison, D.S.O., M.B., Ch.B., F.R.C.P. (E.).

"The Diagnosis and Treatment of Acute or Early Syphilis"—by E. T. Burke, D.S.O., M.B., London.

"The Diagnosis and Treatment of the Local Complications of Gonorrhoea in the Male"—by V. E. Lloyd, M.C., M.B., B.S., Guy's Hospital.

"Gonorrhoea in Women"—by Margaret Rorke, M.B., Ch.B., M.C.O.G.

"Virus Diseases of the External Genitals and Chancroid"—by Robert Lees, M.B., Ch.B., F.R.C.P. (E.), Edinburgh.

"Venereal Disease in Children"—by F. R. Curtis, M.B., Ch.B., M.Sc.

"Cancer of the Rectum"—by Sir Charles Gordon Watson, K.B. (E.), C.M.G., F.R.C.S.

"The Practitioner"—September, 1936.

This number contains a symposium on "Industrial Medicine" comprised of the following articles:

"The Prevention of Disease in Industry"—by John C. Bridge, F.R.C.S. (E.), H.M. Senior Medical Inspector of Factories.

"Physical Standards in Industrial Health"—by Sir David Munro, K.C.B., M.B., F.R.C.S. (E.).

"Industrial Absenteeism"—by N. Howard Mumery, M.R.C.S., L.R.C.P.

"Industrial Poisons"—by Donald Hunter, M.D., F.R.C.P., London Hospital.

"Industrial Diseases of the Lungs"—by W. E. Cooke, M.D., F.R.C.P., D.P.H.

"Psychological Disorders in Industry"—by Millais Culpin, M.D., F.R.C.S.

"Some Practical Aspects of Industrial Hygiene"—by Donald Stewart, M.D., M.R.C.P.

"The Prevention and Cure of Occupational Dermatitis"—by P. B. Mumford, M.D., F.R.C.P., Manchester.

"The Canadian Medical Association Journal"— September, 1936.

"Clinical Experiences with Protamine-Zinc-Insulin and Other Mixtures of Zinc and Insulin Diabetes Mellitus"—by I. M. Rabinovitch, J. S. Foster, A. F. Fowler and A. C. Corcoran.

"The Results of Sympathectomy in Children"—by John L. McDonald, M.B., University of Toronto, Toronto.

"Rheumatic Infection in Childhood: Observations on the Sedimentation Rate and the Schilling Count"—by R. R. Struthers, M.D., and H. L. Bacal, M.D., Montreal.

"The Surgeon's Responsibility in the Treatment of Duodenal Ulcer"—by Roscoe R. Graham, M.B., F.R.C.S. (C.).

"Migraine"—by David Slight, M.B., Ch.B., D.P.M., F.R.C.P. (C.).

"The Prognosis of Coronary Thrombosis"—by G. F. Strong, M.D., Vancouver.

"A Further Report on Obstetric Analgesia and Amnesia"—by Lieighton C. Conn, F.R.C.S. (C.), and John Ross Vant, M.C.O.G., Edmonton.

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"Silicosis Research"—by F. G. Banting, Dept. of Medical Research, Banting Institute, Toronto.

"Silicosis and its Incidence in British Columbia"—by C. H. Vrooman, M.D., C.M., F.R.C.P. (C.), Vancouver.

"The American Journal of Surgery"—September, 1936.

This issue contains a number of articles forming a symposium on Gynaecology among which number are the following excellent articles:

"Amenorrhoea; Menorrhagia; Metrorrhagia; Delayed Menopause"—by H. S. Crossen, M.D., F.A.C.S. and Robert J. Crossen, St. Louis, Mo.

"Endometrial Cycle and Mechanism of Normal Menstruation"—by Somers H. Sturgis, M.D., and Joe V. Mey's M.D., Boston, Mass.

"Dysmenorrhoea"—by Albert Mathiue, M.D., F.A.C.S., Portland, Oregon.

"Sterility"—by Sophia J. Kleegman, M.D., F.A.C.S., New York.

"Operative Treatment of Sterility"—by Francis W. Sovak, M.D., F.A.C.S., New York.

"Pelvic Endometriosis and Its Treatment"—by Emil Novak, M.D., F.A.C.S., Baltimore.

"Vaginal Hysterectomy: Clamp Method for Uterine Prolapse"—by J. W. Kennedy, M.D., F.A.C.S., Philadelphia.

"Le Fort Operation for Uterine Prolapse"—by Fred L. Adair, M.D., F.A.C.S. and Laura D. Sef, M.D., Chicago.

"Treatment of Prolapse of Uterus by the Manchester-Fothergill Operation"—by Charles A. Gordon, M.D., F.A.C.S., Brooklyn, N.Y.

"Cancer of Corpus Uteri"—by William P. Healy, M.D., F.A.C.S., New York, N.Y.

"Fibromyoma Uteri"—by J. P. Greenhill, M.D., F.A.C.S., Chicago.

"Salpingitis"—by J. Randolph Gepfert, M.D., New York.

"Blood Transfusion in Gynaecology"—by R. E. Stetson, M.D., New York.

"The Clinical Journal"—September, 1936.

"The Modern View of Nephritis and its Treatment"—by F. A. Roper, M.A., M.D., M.R.C.P.

"Medical Treatment of Urinary Infections"—by Reginald Ellis, M.D., M.R.C.P.

"Manipulative Surgery"—by T. McW. Millar, M.B., F.R.C.S. (E.).

"Tests for Drunkenness"—by T. H. Blench, M.D., Police Surgeon, Manchester.

"Canadian Public Health Journal"—August, 1936.

"A Recent Outbreak of Haemorrhagic Smallpox in British Columbia"—by J. W. McIntosh, B.A., M.B., D.P.H., Vancouver.

"Progress in Public Health in Canada"—by C. G. Power, B.A., Minister of Pensions and National Health, Canada.

"The Clinical Journal"—August, 1936.

"Haemorrhage in Early Pregnancy"—by A. M. Claye, M.D., F.R.C.S., Leeds.

"The Diagnosis of Some Febrile Conditions"—by G. Lovell Gulland, C.M.G., M.D., F.R.C.P. (Ed.) Edinburgh.

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OBITUARY

DR. JON STEFANSSON

Dr. Jon Stefansson died in the Winnipeg General Hospital on September 29th of pneumonia. He had been present at a celebration at Gimli on September 19th when Lord Tweedsmuir visited the pioneer Icelandic settlement, but was stricken down a day or two later.

His name was internationally known for his operation for the relief of glaucoma. He was born in Iceland, 1878, and at the age of ten came to Manitoba with his father, who settled on a farm at Cypress River. He was educated at Wesley College and the Manitoba Medical College. After an apprenticeship with the late Dr. S. W. Browne, he spent two years in study in London, Glasgow, Vienna and Berlin, narrowly escaping internment at the outbreak of the Great War. On returning to Winnipeg, he carried on practice in diseases of the eye, ear, nose and throat.

He took a keen interest in the welfare of his Icelandic countrymen, read widely, and delighted in music. His wife, Joanna Philipowska, Russian opera singer, predeceased him, but he is survived by a son and daughter.

DR. ROBERT STIRTON THORNTON

Dr. Robert Stirton Thornton, aged seventy-three, died at Vancouver on September 18th. Although the last five years of his life were spent on the Pacific coast, his active life was lived in Manitoba, and on account of his position in the legislature and in the Masonic craft, he was almost as well known in Winnipeg as in Deloraine, where he practised from 1887-1931. He was born at Edinburgh in 1863, was educated at Terriot's College and the University of Edinburgh, and started practice shortly after graduation at Deloraine at a time when settlers were crowding into southern Manitoba. He was Chairman of the School Board at Deloraine for some years and was a prime mover in establishing the park there, which is so well known to visitors. In 1911 he was elected to the Manitoba Legislature, was defeated in 1914, but was again returned in 1918 and served as Minister of Education in the Norris Government. In this capacity he did much to unify education and to stamp out multilingualism. He planned to make the school a community centre. In 1914 he was elected President of the Medical Council of Canada, succeeding Sir Thomas Roddick. In his residency a Dominion-wide license to practice was obtained. Queen's University honored him with the LL.D. in 1914.

The death of his wife, following a motor accident five years ago in which he, himself, was severely injured, affected him greatly.

A man of kindly and congenial disposition, a skillful physician and a statesman of clear vision, Dr. Thornton set his mark on the Canadian West.

DR. WILFRED LORNE ATKINSON

Dr. Wilfred Lorne Atkinson died at his home in Selkirk, Manitoba, on September 15 at the age of fifty. He was born at Clearwater, Manitoba, and graduated in medicine from the Manitoba University in 1911. He practised for nearly twenty-five years in Selkirk and took a leading part in the life of the community, especially in the formation of the Selkirk Golf Club. After the war he served for a time on the staff of the Tuxedo Hospital. A heart condition crippled him during the latter years of his life and to seek relief he took a long sea voyage about two years ago. He is survived by his widow.

DR. GASPARD L. MARSOLAIS

Dr. Gaspard L. Marsolais, St. Eustache, Manitoba, died at St. Boniface Hospital on September 14th. He was born at Joliet, Quebec, in 1873, and came to Manitoba as a youth of nineteen. Graduating in medicine he returned to St. Eustache where he became medical health officer for the municipality. A widow and son survive.

NOTICE

A special program of lectures and demonstrations in medicine and surgery will be held under the direction of The Mayo Foundation from November 9 to 13, inclusive. Mornings will be devoted to surgical and medical clinics. In the afternoons and evenings symposiums will be conducted on neurology, gynecology, diseases of the ear, nose and throat, laboratory procedures, emergency treatment, pediatrics, and renal diseases. In addition, a clinico-pathologic conference will be held. While this program is arranged primarily for the Fellows of the Foundation, visiting physicians are invited to attend.

NOTICE

The Municipal Hospitals of Winnipeg recently purchased a Drinker Respirator for use in various forms of respiratory failure, particularly that associated with Infantile Paralysis. It has also been used for post-operative respiratory failure and the use might be indicated in various other clinical conditions such as various forms of poisoning, etc.

The respirator is available without extra charge for cases treated at the Municipal Hospitals. The respirator is also available for use in suitable cases outside the Municipal Hospitals, provided it is not so far away but that it can be speedily returned if necessary. A nominal charge will be made to help cover the cost of the special transportation insurance when the respirator is loaned out.

Dr. A. B. Alexander, Medical Superintendent of the Municipal Hospitals, would arrange a demonstration for any group of medical men who might be interested.

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